The Examiner has rejected claims 1, 2, and 4 through 11 under 35 U.S.C. §102 in view of Osterrider, or in view of Elbers (either EP 1 129 722 A1 or U.S. Patent 6,703,231). Reconsideration of such rejections is respectfully requested. The disclosure of EP 1 129 722 and US patent No. 6,703,231 are identical, Both belong to the same patent family, claiming priority of EP 0010324.1. EP 1 129 722 disclosure a gM negative EHV-1 virus (H_delta_gM3b1). The genome of this virus is shown in Figure 4 (page 19). This virus was constructed through the substitution of the gM gene by the E.coli LacZ gene. Thus, the gM negative mutant disclosed in EP 1 129 722 and US 6,703,231 comprises heterologous sequences in form of the E.coli lacZ gene. Claim 1 of the subject patent application (claim 2 and 4 – 11 all depend from claim 1) is directed to an equine herpes virus, wherein the protein gM is absent, and wherein said EHV is free of heterologous elements. Thus, subject-matter of claim is not anticipated by the disclosure of EP 1 129 722 and US 6,703,231 under 35 U.S.C. §102.

Osterrider et al., 1996, describes an EHV mutant (L11_delta_gM) also bearing an E.coli LacZ gene inserted into the EHV-1 strain RacL11 gM with characteristics of the parental EHV-1 RacL11. Claim 1 of the present patent application is directed to an equine herpes virus, wherein the protein gM is absent, and wherein said EHV is free of heterologous elements. Thus, subject-matter of claim 1 is not anticipated by the disclosure of Osterrider et al., 1996, under 35 U.S.C. §102

The EHV-1 clone disclosed in US 6,703,231 and EP 1 129 722 is the closed prior art, because this clone is superior over the clone disclosed by Osterrider in 1996. While the Osterrider clone of 1996 shows some residual immunity in respect to gM protein, this was not observed for the EHV-1 H_delta_gM3b1 clone disclosed in US 6,703,231 and EP 1 129 722. Surprisingly, the complete absent of any gM immunity led to a better acceptance of a vaccine comprsing this live modified EHV in horses. Horses vaccinated with the EHV-1 H_delta_gM3b1 clone surprisingly show only a slight weight reduction as compared to horses vaccinated with a gM mutant (e.g. the Osterrider EHV mutant of 1996). However, the EHV-1 H_delta_gM3b1 clone comprises the lacZ gene of E. coli. The problem that was solved by the present invention was finding a gM negative mutant

that does not show any gM immunity, and also producing higher infectivity as compared to the gM negative strains known in the prior art.

The solution of this problem is provided by subject-matter of claim 1. The deletion of the lacZ gene results in a gM negative mutant that is free of heterologous elements. Moreover, it was found that this new mutant shows a higher extracellular infectivity as compared to the gM negative mutant known in the prior art (evidence is given on page 15, 2nd para. and Figure 5 of the specification of the subject application). This result was not expected by or predictable for a person skilled in the art. Moreover, a gM negative EHV-1 clone, that is free of any heterologous elements, is also safer when used as a vaccine.

In view of the foregoing, it is respectfully submitted that the subject application is in condition for allowance and such favorable action at an early date is earnestly solicited.

Respectfully submitted,

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